Briggs (W. T.)

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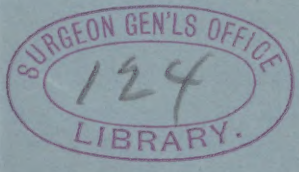

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THE SURGICAL TREATMENT OF EPILEPSY

ARISING FROM INJURIES OF THE HEAD, WITH SPECIAL REFERENCE TO THE USE OF THE TREPHINE.*

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No instance in the history of medicine affords a more brilliant result of experiments on animals, in the elucidation of disease, than those which have been made in the investigation of the essential nature and pathology of epilepsy.

For centuries literally nothing was known of its nature, causes, or treatment. The group of symptoms, however, which characterizes its attacks was so marked and prominent that even the most casual observer could not fail to be impressed. Accurate descriptions of the disease have been transmitted to us from the earliest period. Hippocrates, especially, has in his own inimitable manner drawn a vivid and lucid pen-picture of the symptoms of the disease. In the absence of physiological knowledge and of pathological data from which to form an idea of the essential nature of epilepsy, it is not wonderful that no progress beyond the observation of its coarser manifestations should have been made, notwithstanding the many efforts of the most brilliant minds to elucidate its nature or the various theories advanced to explain its phenomena.

For centuries not a single ray of light was shed on the subject by which the impenetrable darkness surrounding it might be dispelled.

Marshall Hall, by his experiments on animals, was the first to illumine this hitherto obscure subject and to blaze a path in

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the pathological wilderness by which all investigators might be guided. In 1850 he made a full and explicit enunciation of his theory of the reflex action of the nervous system (Croonian Lectures, delivered at Royal College of Physicians, London), and by his clear and lucid explanation enchained the attention of the profession over the whole world. His theories of the reflex action of the nervous system are now universally received and acknowledged. He pointed to the medulla oblongata as the center of the reflex nervous system, and proclaimed that all paroxysmal seizures were one and the same, differing only in degree; that the causes of these paroxysmal seizures were centric and eccentric; that these causes act on the reflex nerve center; that this center exhausts itself upon being called into powerful and frequent action, but gradually recovers, thus accounting for the cessation and recurrence of the seizures; and he especially claimed that epileptic attacks, like every reflex action, were always excited.

The name of this great philosopher and physiologist will ever be remembered, when the subject of nervous diseases is discussed, as the pioneer in that important and difficult branch of medicine—as the first to point out the way in which inquiries should be prosecuted. He gave such an impetus to the investigation of physiological and pathological studies that other energetic and enthusiastic investigators speedily followed.

Brown-Séquard, one of the most indefatigable workers in experimental physiology and pathology, after he had for twenty-five years investigated epileptic convulsions artificially produced in guinea-pigs, demonstrated that convulsions resembling epilepsy were generally developed by wounding various portions of the nervous system; that in a short time after such injuries a state of increased irritability was developed and the epileptogenic zone, characterized by a certain degree of hyperesthesia of the cheek and the antero-lateral regions of the neck, was formed, and that slight irritations of this region, by pinching the skin or pulling the hair, were sufficient to produce attacks. His conclusions are that the attacks are alone dependent upon a

peculiar irritation, starting from the cutaneous ramifications of some centripetal nerves, and that even when they have their primitive cause in the nervous centers, some cutaneous nerve filaments are brought into a state of irritation and thus excite the attack.

The views of these investigators have, in the main, been confirmed and sustained by the researches of Schroeder Van der Kolk. According to this pathologist, epileptic convulsions are caused by an exalted action of the ganglionic cells of the medulla oblongata which are more or less rapidly exhausted by the spasms. The irritability of the cells having been restored by rest and nourishment, the capacity for reflex action is regained, and, upon the application of an external irritant, convulsions again ensue. He is of the opinion that though the primary irritation might be remote, a morbidly elevated sensibility of the medulla oblongata is always present and is the essential cause of the attack, rendering the organ mentioned more capable of discharging itself in involuntary reflex movements.

Nothnagel has confirmed the opinion of Van der Kolk by proving, experimentally, that there is a convulsive center in the medulla oblongata, a circumscribed spot through which general convulsions may be induced by reflex excitation. The medulla oblongata is also the seat of the vaso-motor center, the respiratory centers, etc., which centers are always more or less affected in epileptic seizures. It is, therefore, reasonable to conclude that the medulla oblongata is the chief seat of the disease. This theory harmonizes the physiological principle with the pathological data, and it is now well understood that peripheral irritation conveyed to a susceptible medulla, whether from another part of the nervous system, from some viscus, or from a cutaneous nerve, equally excites epileptic convulsions.

Although the malady is characterized by symptoms clearly referable to certain parts of the nervous centers, no marked physical changes of those parts are discoverable after death. It is true that Van der Kolk, by microscopical examinations of

the pons and medulla of epileptics, discovered certain changes, to wit, enlargement of the vessels, particularly in the posterior part of the medulla, together with an albuminous exudation between the nerve fibers, leading to induration, then to fatty degeneration and softening. Echeverria, by his post-mortem examinations and experiments, confirmed the opinion expressed by Van der Kolk. Myers also discovered a diseased condition of the blood-vessels, fatty degeneration and sclerosis, not only of the medulla, but also of the upper part of the spinal cord and of the cerebellum. There is scarcely a disease or injury of the cranium or its contents which has not been noticed in connection with the disease, but these various conditions have often been found in the bodies of persons who died of other diseases, and who had never been the subjects of epileptic convulsions. Again, examination of those parts in epileptics who had died from intercurrent diseases have revealed not the slightest changes. It is a fact fully authenticated, that no invariable lesion of the nervous centers, or of any other part, has been determined on as the *causa proxima* of epilepsy. It is probable that various pathological changes in the brain or its membranes, or in the nerves, whether sensor, motor, or sympathetic, may excite the parts of the pons and medulla in which the reflex cells are located and give rise to the group of symptoms which characterizes epilepsy, just as a sensory nerve always gives expression of pain and different impressions. No discoverable pathological lesion is present in the earlier stages of the disease, and those found in the post-mortem examinations of epileptics are secondary or accidental. Epileptics seldom die from the disease, and although attacks may be of frequent occurrence and of great severity, they may live for many years in the enjoyment of perfect health, and are able to attend to the most weighty duties of life. History tells us of a number of the most gifted men, whose achievements with pen or with sword stamped them as individually great, who were the subjects of frequent attacks of epilepsy.

Epilepsy should then, in my opinion, be regarded as essen-

tially a functional disease; for, while it is generally conceded that every vital act is followed by some change in the organism and every diseased condition must necessarily be accompanied with change of tissue, science has not yet been able to demonstrate those changes.

The origin of the disease is doubtless due to an increased excitability of the medulla oblongata and pons varolii—the reflex nerve centers—in consequence of which these reflex centers respond to peripheral irritation much more readily than in their normal condition, and the motor manifestations, although they do not differ in their nature from the normal reflex movements, are intensified far beyond their normal degree.

The cells of the excito-motor and vaso-motor centers are so highly charged with excitability that they explode on the application of a stimulant conveyed through the nerves. This part of the central nervous system has been aptly compared to a Leyden jar, which explodes upon the contact of an electrode when sufficient tension has been attained (Van der Kolk). The state of tension in the medulla oblongata is gradually induced until it reaches its acme, when from the action of some peripheral irritant, spasm succeeds spasm, until the excitability is exhausted. It again accumulates, to be again discharged in the well-known routine of the epileptic cycle.

The phenomena of an epileptic seizure are due, in the first instance, to excitation of the vaso-motor center which induces constriction and consequent anemia of the cerebral vessels, causing the loss of consciousness from the abolition of the activity of the cortex cerebri. Often this is the only phenomenon met with, and it then constitutes what is known as *petit mal.* But in a larger number of cases the excitation of the *convulsive center* comes on simultaneously and gives rise to general convulsions. The spasm of the cerebral vessels, as well as those of the face, is of very short duration, and is rapidly succeeded by paralysis and venous congestion of those vessels, which is greatly aided, if not caused, by the spasmodic contraction of the muscles of the neck. The interference with the respiratory act and

the intense venous congestion of the cerebrum readily account for the coma succeeding the convulsion.

The experiments of Kussmaul and Tenner prove that cerebral anemia stands in definite relationship to epileptic seizures. Donders made direct examination of the brain by means of a watch-glass placed in the opening of a trephined skull, and demonstrated experimentally the anemic condition of the brain. Others have contended that epilepsy depends upon cerebral hyperemia. Like most contested points, there is truth on both sides of the question. There is, doubtless, in the commencement of every attack, upon the discharge of the vaso-motor center, a spasm with consequent anemia of the cerebral vessels, but this effect is momentary, and is succeeded rapidly by intense venous congestion.

A case in which I resorted to the trephine for the relief of traumatic epilepsy, in 1878, illustrated in an admirable manner the changes that take place in the intra-cranial vessels during a convulsion. A spur of bone from the inner surface of the button, which was situated on the top of the head directly over the sagittal suture, had perforated the dura mater and entered the longitudinal sinus. As soon as the bone was removed from its position by the trephine a continuous stream of blood followed. A soft sponge was at once pushed into the opening for the purpose of arresting the flow. A violent convulsion was immediately excited when the sponge was removed. For perhaps half a minute the face became very pallid and the flow of blood from the opening in the sinus ceased entirely; then the face became purple, and the blood gushed forth in a large stream with great force. The sponge was again pressed into the opening, with the effect of restraining the hemorrhage, and retained in position with a bandage. The patient recovered without further trouble.

It will not be denied, I think, that *two factors* are essentially necessary in the development of every epileptic seizure. First, the increased and persistent excitability of the reflex centers, and, second, the peripheral irritant. The disease can not occur

without the concurrence of both of these factors. An individual may have all the predisposing circumstances necessary to place the reflex centers in the highest degree of susceptibility, and this susceptibility may lie dormant for years, even for a lifetime, unless the exciting cause comes into action. It is true that in many cases no peripheral irritant can be discovered, and oftentimes even the influences which develop the increased excitability of the reflex centers are not manifest. That the exciting cause can not be ascertained is no argument against the existence of such. In a great number of diseases the causes acting in their production are entirely unknown. It has been aptly said, "Happy is the physician who knows the cause of disease," for most usually when the cause becomes known its essential nature and treatment are easily discovered.

Inherited nervousness is the most frequent cause of the exaggerated excitability of the reflex centers. It is not essential that the parents should have had epilepsy to transmit the peculiar susceptibility to the offspring. It is only necessary that the father or mother, or some ancestor, should have been cataleptic, neuralgic, or the possessor of a very irritable nervous system. The irritable sensitive medulla and pons varolii are inherited, not the epilepsy.

The nervous erethism engendered during dentition and at the period of puberty often develops the peculiar predisposition. So also will the long-continued use of alcoholic stimulants, sexual excesses, syphilis, and, in fact, any cause which is calculated to enfeeble the general system or to induce hyper-sensibility of the nervous system. Probably no circumstance more frequently predisposes the reflex centers to abnormal discharges than powerful and persistent local irritations. The constant fretting of these centers is almost certain to develop the necessary excitability; besides, it has been shown by the experiments of Tiesler, Klemm, Niedieck, and others, that these local irritations often set up ascending neuritis in the peripheral nerves which gradually approaches the central nerve mass, involving it in the same diseased action. Hayem says the path of transmission of the

irritation from the nerve to the spinal center is two-fold—first, by the interstitial tissue; and secondly, by the axis cylinders which are swollen and moniliform in the central end of the nerve.

The exciting causes of epilepsy, which are supposed to exercise a more direct and manifest influence in the production of the disease, are as numerous as the circumstances which may give rise to irritation in various parts of the body, as teething, intestinal worms, indigestible food, disorders of menstruation, genital irritations, neuromata, lesions of nerves, retraction of cicatrices, injuries of the skull, foreign bodies, etc. I wish, however, to restrict my remarks to epilepsy arising from injuries of the head, which demand the consideration of operative measures.

Echeverria estimates that full ten per cent of all epileptic seizures are due to injuries of the head. Such wounds are more likely to excite attacks than those of any other part of the body, for the reason, probably, that the branches of the trigeminus nerve, which has its origin directly from the reflex nerve mass, are freely distributed to parts of the scalp, and to every part of the dura mater; peripheral irritations are therefore more readily transmitted; the flame is much nearer the combustible. Even when the primary effects of such wounds have been dissipated, or when they were too slight to have claimed attention, they are not infrequently, sooner or later, followed by the most distressing attacks. While it is true that great violence is often inflicted on various parts of the head without the slightest disturbance of the nervous system, it is equally true that epileptic attacks often occur from injuries of the most insignificant character. Westphal has proven conclusively by experimentation that epileptic convulsions, recurring every two or three weeks, may be produced by slight blows on the heads of guinea-pigs. A case illustrative of this fact came under my observation in the human subject a few years since. A bright lad, twelve years of age, who had no hereditary predispositions, while running a foot-race with some companions, struck the right side

of his head against a tree, producing a contused wound of the scalp, just above the lower border of the parietal bone. A short time afterward he was seized with a convulsion. He had a recurrence of the spasms in three weeks much more severe than the first, and they have since continued to recur at much shorter intervals and with greater violence, till now he is in a state of confirmed fatuity. I was consulted by the father in reference to the use of the trephine in this case, but after mature consideration I became satisfied that the indications for the operation were not sufficiently marked, and therefore declined interference. He afterward fell into the hands of other surgeons, who did trephine the skull, but with no benefit.

It is impossible to predict the consequences of violence inflicted on the head, however severe its nature or however trivial its morbid manifestations. It is equally difficult to foretell the time when the evil results will take place. It may be in a few hours or days, or it may be postponed for twenty years or longer. Professor Dudley used to mention in his lectures a case in which epilepsy resulted from an injury of the skull, inflicted sixteen years before, which was entirely cured by the trephine.

Several cases have come under my observation in which ten years elapsed before the occurrence of the attacks.

The trauma may involve merely the scalp in a contused or lacerated wound, which, in healing, forms a firm, hard cicatrix, entangling some of the terminal extremities of the nerves, thus setting up a peripheral irritation that in course of time excites an attack of epilepsy. Liddel, in a very interesting paper, published in the Transactions of the American Neurological Association (vol. i, p. 157, 1875), calls attention to the fact that the entangling of nervous fibrillæ in cicatrices of wounds gives rise to neuralgia and other neuroses. The same condition may excite epileptic attacks in persons who possess the necessary susceptibility. Five cases of epilepsy have come under my observation which could certainly be traced to this condition as a cause. In each of these cases the cicatrices were the seat of pain and of a burning sensation which occurred at

irregular intervals, lasting for a few days, then disappearing for a time. Increased sensibility, together with elevated temperature of the part involved, were constantly present. The morbid sensibility was so acute in several of the cases that the slightest contact with the seat of the original wound gave rise to great suffering, and in one case pressure upon or even handling the part in the necessary examination was followed by an attack of epilepsy within the succeeding twenty-four hours. In every case the patient was gloomy, despondent, and listless, irritable, irascible, and disinclined to any kind of business. The time which elapsed from the reception of the wound to the first epileptic seizure varied from two months to three years.

A blow on the head, whether the scalp is lacerated or not, may result in serious mischief to the bones of the cranium without fracture. Pericranitis may be developed, with subsequent disease of the bones, or the bones may suffer violent contusion at the time of the injury. In either case the diseased action is very likely to be followed by osteo-sclerosis, or by hypertrophy from the persistence of hyperplastic inflammation. The eburnation or the hypertrophy is most usually diffused, but is sometimes localized and confined to a small surface. I have met with several cases in which the eburnation involved but a very limited portion of bone, and others in which the hypertrophy was exhibited in the formation of small nodules of bones, some not larger than a garden pea, others as large as an almond. In two cases which have fallen under my observation there were narrow spurs of bone growing from the inner surface of the calvarium nearly one half an inch in length.

When the eburnation or the hypertrophy is localized, epilepsy is much more liable to be excited than when they are diffused. The effect of pressure by points upon the brain or its membranes never fails to give rise, sooner or later, to the most serious neurotic affections. The presence of a point of irritation caused by such changes in the bones is indicated by pain, a sense of pressure or weight, or of soreness in the injured parts,

together with headache, and at times with symptoms referable to disturbance of the motor area of the brain.

Again, the inflammation may be of the destructive variety, and lead to necrosis or osteo-porosis, which are followed by the same symptoms that attend those pathological conditions in other parts of the body. It is not difficult to understand these pathological changes in the cranial bones from blows upon the head, when we reflect, in the first place, that the relationship is so close, the association so intimate between the pericranium and the dura mater that a morbid state of the one very rarely exists without implicating the other, and in the second place, that the coverings of the brain, the tutamina cerebri, are composed of so many histological elements, which differ widely in their capacity for healing. The common integument is more vascular, more sensitive, and enjoys in a higher degree the power of restoration than the parts which it covers, so that while it may have quickly recovered from the effects of a wound, the parts beneath are still laboring under the influence of the injury, and the changes mentioned may result from perverted nutrition or from pathological action.

But the most frequent causes of traumatic epilepsy are old fractures of the cranial bones; fractures which may have been produced months or years previously. The brain is protected against injury by a bony spheroid which is accurately adapted to its surface, so that any irregularity of the internal surface of the bony envelope produced by fracture will certainly impinge to a greater or less extent upon the membranes of the brain. The point of pressure will in many cases give rise to meningeal and cerebral irritation associated with symptoms of concussion or compression. Unless serious injury has been inflicted at the same time upon the brain or its membranes, these symptoms will pass off without any aid from the surgeon. The irritation, however, characterized by its special symptoms, will in many cases persist and demand the careful consideration of surgical means for its relief. The sentiment of the mass of the profession has been and still is in favor of non-interference in most fractures of the cranial

bones with depression, unless attended with marked and persistent symptoms of compression of the brain. An exception, however, is made by the majority of surgeons in punctured fractures, and by some in compound. It is true, that when the inner surface of the depressed portion of bone is smooth and broad, the brain will readily accommodate itself to the pressure, however great it may be, and the primary symptoms will soon pass off; but when greately depressed fragments of sharp, spiculated bone are driven upon, probably into, the cerebral mass, cerebral and meningeal irritation will speedily ensue and often run into fatal encephalitis. Under these circumstances the early resort to the trephine is, in my opinion, demanded to prevent the immediate consequences as well as to guard against the remote effects. If the trephine is withheld under these circumstances and the expectant treatment adopted in lieu, the patient may escape immediate death, but will live in constant jeopardy from the slow, insidious form of irritation which will often call for redress at some remote period. Even when the injury to the intra-cranial contents is not so great as to produce immediate effects, the slight point of irritation left will often sooner or later excite epileptic attacks. Again, it frequently happens that in the repair of broken fragments of the skull, osteophytes spring from the new bone formation which fills the interspaces between the fragments, and the pressure of their sharp points upon the membranes evokes the necessary amount of irritation for the development of the disease. Preternatural pressure upon the membranes of the brain may be borne without much evidence of its existence for a time, but if long continued will give rise to such irritation and increased sensibility at the point of pressure that bad results will almost inevitably follow. A person who is the subject of accentuated pressure upon the membranes of the brain from fractures of the skull is like one who has the sword of Damocles suspended over his head, which is liable at any time to fall with disastrous effect.

The inveterate nature of this distressing and terrible disease, and the doubt pervading the professional mind since the days

of Hippocrates and Galen as to its entire and permanent curability, have caused epilepsy to be regarded as a reproach upon our art. An attack of the severer type of the disease overwhelms the subject and the friends with consternation, and its repetition, often frequent, robs them of confidence or hope of relief, while the display of its ensign to the physician and surgeon is a signal for a contest in which trustful and assured reliance in the resources of therapeutics or operative endeavor is either absent or greatly weakened. The history of the prognosis of the disease, as held by the great lights of our science, presents a varying view, some claiming a large majority of cases to be hopeless, while others of equal authority have with more patience and accuracy distinguished its types of different origin and causation, and have reached more favorable and auspicious conclusions. Under advancing knowledge of its essential pathology, the number of the latter is steadily increasing, and with reference to its treatment the appliances of the therapeutic armament are becoming more full and scientific, as well as satisfactory in results. The pathological postulate advanced in this paper is, that epilepsy is fundamentally reflex in its character, and this can not be challenged as to many expressions of the type known as epilepsia gravior. The opinion is herein ventured that the prognosis in those cases which are distinctly reflex, and which undeniably have their origin in eccentric irritations, under prompt and judicious treatment should be favorable; and that the removal and abatement of the irritant cause threatening or inducing the dire consequences of the disease is a rational and scientific procedure, in the highest degree justifiable in the hands of the discriminating surgeon. There can be no doubt but that the timely and skillful removal of the traumatic sources of peripheral irritation will yet relieve the profession of the pessimistic prognosis with which it has so long deemed it necessary to enshroud the hapless victim of epilepsy.

It has already been claimed that *two factors* are essential to every epileptic attack, viz., increased susceptibility of the reflex nerve centers and peripheral irritations, and that these conditions must be in operation at the time of the attack.

In November, 1879, a lad, seventeen or eighteen years of age, was received in our hospital suffering from frequent and violent attacks of epilepsy, which followed an injury inflicted on the head in a fall from a stable-loft some four or five years previous. The convulsions commenced about a year after the injury, and recurred at first once a month, but had gradually become more frequent, until he came into the hospital. He then had returns every week, and as many as fifteen or twenty spasms during the day and night. The day after the attacks he usually got up and was apparently as well as ever with the exception of general soreness.

The seat of the injury, which was found on the forehead near the edge of the hair, exhibited a well-marked scar of the scalp, with a slight depression of the bone underneath. Constant pain and tenderness, greatly increased by the approach of the convulsions, were present. He was brought into the amphitheater of the hospital and examined by Professors Choppin, of New Orleans, Stephen Smith, of New York, Mussey, of Cincinnati, L. P. Yandell, of Louisville, and other distinguished gentlemen, delegates to the Health Association which was then in session in our city. Without a dissenting voice they concurred with me in the opinion that the trephine was not only indicated but urgently demanded.

The trephine was used in the presence and with the kind assistance of the gentlemen mentioned; the portions of bone removed exhibited small osteophitic growths along the line of the original fracture. The dura mater was apparently healthy. The wound was left open for free drainage. A short time after the patient was carried to the ward he had a severe convulsion, but this was the last he ever had. His wound healed kindly, and he continued to improve for more than two months. At this time it was noticed that he walked unsteadily and complained of vertigo. The unsteadiness of gait increased so that in a short time he reeled like a drunken man when he attempted to walk. His respiration became irregular and deglutition difficult, and at the end of five months he sank into a deep lethargy which ended in death.

A post-mortem examination revealed the medulla oblongata and pons very much softened, with greatly enlarged vessels, the pathological changes extending into the under surface of the cerebellum. In this case the removal of the peripheral cause put a stop to the oft-repeating spasms, although the medulla and pons were in that special condition which is most likely to give rise to the disease, and progressed to the death of the patient without a symptom of the return of the epilepsy.

The indication, then, to be observed in the treatment of the disease is to sever the combined action of these two factors; to break up the inter-dependence existing between them by (1) removing the existing cause, the peripheral irritation, and (2) by modifying or subduing the morbid excitability of the reflex center. In attempting to carry out the first indication of treatment in the cases to which my remarks are especially addressed, those arising from injuries of the head, too much care and attention can not be bestowed on the subject. Each individual case should be investigated with the view of determining the exact nature and situation of the exciting cause, of tracing out the source of the peripheral irritation.

It frequently happens that old injuries of the head, even when associated with epilepsy, have no causative effect in the production of the attacks—that they are merely incidental, while the true exciting cause is situated in some remote part of the body. A little girl from McMinnville, Tenn., twelve years of age, was brought to me, who had four years previously fallen from a porch, a distance of nine feet, and injured her head, as well as the left leg between the knee and ankle. She was rendered insensible for several hours, but had no further trouble from the head injury, which healed kindly and rapidly. Her leg, however, continued in an inflamed condition, and eventually an abscess formed and was opened. In the course of a few months a number of fistulous orifices opened, through which a probe could be passed to denuded bone. Two years subsequent to the fall she was attacked with epilepsy, which continued to recur every few weeks until she was brought to the city and

placed in my care. Upon examination a well-marked depression of the skull was discovered just above the anterior-inferior angle of the parietal bone. There were, however, no symptoms indicating a point of irritation connected with it. There was neither pain nor increased sensibility, nor elevated temperature, nor other evidences of trouble about the part involved in the original head wound. The skin of the leg about the injured region was of a dark livid hue, and was perforated with a number of fistulous openings, through which the probe detected dead bone. The limb was the seat of continuous pain and increased sensibility, which were always greatly aggravated upon the approach of an epileptic seizure. An operation for the removal of the dead bone was made. She has never had a convulsion since, and is now, five years afterward, in the enjoyment of the most excellent health.

In this case, though there had been a fracture of the skull, with well-marked depression of bone, no symptoms of irritation at the seat of the injury of the skull existed. On the other hand, a peripheral irritation was kept up by the necrosed bone of the leg which culminated in the development of the epilepsy.

If the epilepsy is dependent upon the entanglement of nervous fibrillæ in cicatrices of the scalp, and the symptoms (increased sensibility, pain, elevated local temperature, etc.) point to the original wound as the source of the irritation to which the attacks are due, it is the imperative duty of the surgeon to excise the old scar, with its included fibrillæ of nerves, and thus abate one of the factors which is essential to the continuance of the disease. No other remedy offers any hope of relief. In the five cases of this character which have fallen under my observation, the cicatrix was thoroughly removed and the wound left open to heal by granulation. In all of these patients the attacks were arrested. Two have been recently examined, after three years' time.

In the case of a gentleman from Mississippi, twenty-three years of age, the injury was inflicted on the forehead, extending from the edge of the eyebrow to the coronal suture, by a piece of wood, driven off while splitting fire-wood. A firm, indurated

cicatrix marked the site of the original wound. The scar was the seat of constant pain and was very tender to the touch, the most sensitive part corresponding to the course of the supra-orbital nerve. The epileptogenic zone was well marked. He had been the subject of epilepsy for three years and had taken all the usual anti-epileptic remedies. The first attack came on eight months after the injury and recurred every two weeks, preceded by increased local pain, severe headache, etc. The whole of the scar was carefully dissected out, the incision extending downward so as to include the supra-orbital nerve from its passage through the supra-orbital foramen to as near its terminal branches as possible. The night following the operation he had two severe convulsions. Since then he has been entirely free from the attacks, now three years.

In May, 1880, while attending the meeting of the American Medical Association, in Atlanta, I was consulted by a gentleman from Alabama, aged forty-five years, who had been suffering from epilepsy for more than a year, arising, as his physician thought, from an injury to the skull inflicted by robbers with a rough stick. The scalp was severely lacerated and the patient struck insensible, in which condition he remained for a number of hours. He gradually recovered from his stupor and managed to reach his home, when his wound was dressed by his physician. The wound healed but left a very tender, irritable scar. Convulsions followed six months after the injury and recurred sometimes once and sometimes twice a month. More than a year had elapsed and he was still the subject of frequent attacks of epilepsy. Upon examination, a painful, sensitive, and puffy cicatrix was found situated over the junction of the frontal and parietal bones of the left side. The general health of the patient was indifferent. He was feeble, nervous, and sleepless with indigestion, constipated bowels, etc. With the kind assistance of Prof. Gunn, of Chicago, Prof. Lankford, of St. Louis, and others, I excised the tender and sensitive scar to the bone, the surface of which was roughened and irregular. It was the opinion of Prof. Gunn that the trephine should have been used, to

which opinion I was myself inclined; but having no instruments except those of a pocket-case, I had to content myself with what was done. The patient quickly recovered from the operation and returned to his home. I received letters from him frequently for two years. He had no more convulsions, but his health remained bad and he continued very despondent.

It may be safely concluded that when the symptoms point to an old scalp-wound as the source of the eccentric irritation which gives rise to the epileptic attacks, the indication is clear and unmistakable, to excise the cicatrix with its included fibrillæ of nerves.

If the epilepsy is the sequence of a contusion of the cranial bones which has caused hyperplastic inflammation resulting in osteo-sclerosis or hypertrophy, and there are unmistakable evidences of a peripheral point of irritation from the pressure of thickened and irregular bone, or from intra-cranial effusion induced by the diseased action, the trephine may be resorted to with the greatest hope of success. The sclerosis or hypertrophy is sometimes circumscribed and limited to a small point, an exostosis or nodule growing from the inner surface of the skull, at the seat of the original wound, which may be included and removed by the single application of the trephine. Even when these changed conditions are more or less diffused, a special point of irritation may be present, indicated by pain of a dull, heavy character, which, if not constant, always occurs at the same point. The removal of the bone covering this fixed point of irritation will usually arrest the paroxysms of epilepsy.

In other cases many applications of the trephine are necessary to relieve the pressure of the ingrowing bone. In one case in my own practice six large crowns of the trephine were found necessary to surround and separate the thickened and roughened bone, which, after the angles were rounded off with Hey's saw, left an opening as large as the palm of the hand. Yet the patient recovered without an unpleasant symptom, and was cured of his epilepsy.

In those cases which can be referred to the irritation caused

by necrosed or carious bone there are present, in addition to the signs of local irritation, the usual symptoms which characterize the presence of diseased bone. With these symptoms pointing to the origin of the epilepsy, the trephine is urgently demanded, and should be resorted to without hesitation. The operation should accomplish the removal of the whole of the diseased bone, however extensive. In one case, Dr. Stephen Smith removed the whole of the right parietal with a great part of the frontal and occipital bones, which were in a state of caries, the result of a traumatism. Professor Gross reports a case in which a portion of necrosed bone was incarcerated by an overlapping ledge of bone. The sequestrum was not only prevented from escaping in consequence of the narrow opening, but the irritation which its presence exerted upon the brain and its membranes caused repeated attacks of epilepsy, which permanently disappeared upon the extraction of the offending substance. (Gross's Surgery, 1882, p. 88.)

Two very interesting cases of epilepsy, due to necrosis of the cranial bones, have come under my observation:

1. A steamboat captain, thirty years of age, who had been for several years the subject of constitutional syphilis, was struck with a walking-stick on the left side of the upper part of the frontal bone. The wound inflamed, and in a few months the skin covering the part was perforated by a number of fistulous openings which communicated with the dead bone. Eight months afterward epilepsy set in and continued to recur with increasing severity and frequency for nearly a year afterward, when he came under my care. The symptoms pointed clearly to the necrosed bone as the exciting cause of the convulsions. The whole of the dead bone, the size of a silver dollar, was removed by the trephine and Hey's saw. He had two convulsions the day following the operation, but recovered entirely, and has remained free from epileptic attacks.

2. In the second case, which was reported in the Nashville Journal of Medicine and Surgery, 1866, a young man twenty-three years of age was wounded in the battle of Resaca, Ga., by

a minnie ball in the left temporal bone, just above the ear. He was carried to the hospital and examined by the surgeon in charge, who pronounced it a mere scalp wound, and ordered cold water applications. The patient was confined to his bed for a week with fever, but gradually recovered sufficiently to act as a ward nurse. He served in that capacity but a short time, as his mind became more and more feeble, until he could no longer recognize his own friends. He was then discharged from the service and carried to his father's home in Jackson County, Tennessee. There he remained in a state of imbecility for a year. Four months after his return home he was seized with severe convulsions, which recurred two or three times a week. Eight months after the development of epilepsy he was brought to Nashville and placed in my charge, the most deplorable case I ever witnessed, a driveling imbecile, with almost daily convulsions, and incapable of attending to the calls of nature. An examination of the site of the wound revealed a large linear cicatrix an inch above and a little in front of the left ear; about an inch and a half posterior to the ear and on a line with the scar a small pouting orifice was found, giving exit to pus. The probe passed into the small opening came at once in contact with dead bone. With very little hope of relief from the desperate condition in which the patient was placed, I proposed to remove the diseased bone with the trephine, which proposition was eagerly accepted by the friends of the young man. An oval flap was raised and a large-sized trephine applied. In a very few turns of the instrument the bone was cut through and removed. To my utter astonishment, immediately beneath and embraced by a few fibers of the dura mater *was an ounce minnie ball*, which was quickly turned from its bed. The patient recovered after a pretty sharp attack of erysipelas of the scalp. He had a few light convulsions after the operation, when they ceased to return. His health and the condition of his mind gradually improved, but it was six months before he was able to converse intelligently, or to attend to his own wants. He has never been the bright boy he was before he received the wound.

In a conversation with him three years since, he told me that the last thing he recollected was when he was in the battle and the balls were flying so thick and his comrades falling so fast around him that he pulled his slouch hat down over his ears and eyes that he might not hear the unpleasant whistle of the balls or see his comrades fall. His life was a blank to him until several months after the operation.

If the epileptic attacks are referable to marked depression of the bone or to the formation of osteophytes or other changes the result of old fractures of the skull, and the symptoms point clearly to the original wound as the seat of the peripheral irritation, the trephine should be appealed to without the slightest hesitation at the earliest moment possible and the sources of local irritation removed. In such cases the judicious use of the instrument is capable of effecting great and lasting benefit to patients and honor to the profession.

The practice of trephining the skull for the relief of epilepsy was resorted to by the old surgeons without discrimination and without reference to any special indication. Such reckless abuse of the operation caused it to fall into disrepute from which it has never fully recovered. Prof. Dudley, of Lexington, Ky., was the first in this country and possibly the first in the world to advocate and practice the operation from a rational standpoint. The brilliant results which he obtained from the use of the trephine in cases of epilepsy caused by injuries of the head arrested the attention not only of his pupils, who were scattered all over the country, but also of the profession generally. Many American surgeons have since resorted to the operation for the cure of certain forms of epilepsy, among whom may be mentioned Warren, Hayward, Brainard, Dawson, Bigelow, Gross, Pancoast, Gilmore, Van Buren, Echeverria, Blackman Sayre and others. From tables of the operation collated by Stephen Smith, Billings and Echeverria, ninety-two (92) American operations are reported, of which sixty-three (63) were cured, thirteen (13) ameliorated, two (2) not changed, fourteen died. Walsham, in the Bartholomew Hospital Reports of 1883, has collected eighty-

two (82) cases, to which he has added forty-eight (48) collected by Billings and others, making one hundred and thirty (130) in all, of which number seventy-five (75) were completely cured, eighteen (18) improved, seven (7) unimproved or worse, thirty (30) died. To this table may be added thirty (30) cases of epilepsy from old injuries of the head operated upon by myself. Of these, twenty-five (25) were cured, three (3) ameliorated, one (1) not changed, one (1) died.

These statistics prove that the mortality following the operation of trephining the skull in traumatic epilepsy is not greater than that following other operations of equal magnitude. The hope may be reasonably indulged, from the great advances made during the last decade in the treatment of operation wounds, that the mortality in cases involving surgical interference in diseases and injuries of the skull, of the meninges, and of the brain itself may be still further reduced, and that the same improvements in the results of such operations may obtain as in those involving the peritoneum, which was formerly supposed to be as susceptible of rapidly fatal inflammations from traumatisms as the cerebral meninges.

That the operation may be followed by the full measure of success, it is essentially necessary that certain points of practice in connection with it should be thoroughly understood :

1. That the operation should be done as early as possible, because the long-continued irritation and interference with the healthy nutrition of the brain and nervous system will often beget a permanent impression, which will remain after the point of irritating bone has been removed.

2. The operation should be thorough and complete, for if all of the offending part is not removed no benefit will accrue.

3. The operation should be carefully and cautiously done. No one can foretell the changes that may have taken place in the parts around the injured bone, and a rude operation may cause irreparable mischief, which might have been avoided by one carefully planned and skillfully executed.

4. The wound should be dressed after the open method, or

such arrangements made for drainage as will thoroughly effect the object, for a large amount of fluid is discharged from its surface, which, if confined, might cause undue pressure on the membranes and the brain.

5. The most careful after-treatment should be instituted, to prevent destructive inflammation.

With a strict observance of these precautionary measures, I think it may be claimed that the use of the trephine for the removal of points of irritation of the skull which are provocative of epilepsy is as legitimate as lithotomy for the relief of vesical irritation.

The removal of the irritating point of bone with the trephine will often cure the epileptic seizures permanently, without further attention, especially in those cases in which the peripheral irritation has originated and maintained the increased susceptibility of the reflex centers.

Dr. Dudley reported the case of young Goforth who, after he had been cured of epilepsy with the trephine, made many trips to New Orleans on flatboats and always walked back to his home. He never had a recurrence of his convulsions, notwithstanding the great exposure and fatigue to which he was subjected.

In 1862 a Confederate soldier received a fracture of the left parietal bone from a stone thrown by an infuriated farmer in western Virginia. He recovered from the immediate effects of the blow, but in five months afterward he was seized with epileptic convulsions which continued to recur for eight months, when he came to Nashville and entered the hospital for the purpose of submitting to an operation for relief. Upon examination the seat of the original wound exhibited a deep scar of the scalp and a well-marked depression of the bone. Constant pain and soreness were present at the point of injury, and the general health had become much impaired. The trephine was resorted to and the depressed bone removed. The patient never went to bed, but was well in a week. He returned to his home and spent a few months in comparative retirement, but soon becoming rest-

less, organized a guerrilla band, and went into every species of excitement. He killed an old man in cold blood, was arrested by the Federal authorities, tried for murder, and sentenced to be hung. Before he was executed he effected his escape, and singularly enough, went to his old haunts, organized another guerrilla band, committed all sorts of depredations on friend and foe, was at last surprised by a company of Federal cavalry, and after a desperate fight was killed. Yet during all the excitements incident to such a life he had no return of convulsions.

In some cases, appropriate and judicious treatment after the use of the trephine will effect a cure when the treatment without it would have been of no avail. It should not, however, be forgotten that one of the factors of the disease, viz., exalted susceptibility of the reflex centers, often continues in operation after the exciting point of irritation has been removed by the trephine, ready to be excited into activity by some other peripheral irritant. It is, therefore, the imperative duty of the surgeon to institute and maintain, after the operation of trephining for the relief of traumatic epilepsy, such a line of treatment as will in his judgment subdue the exaggerated excitability of the reflex centers and thus destroy the last link in the chain of morbid action.